



Meningococcal Disease in US Military Personnel Before and After Adoption of Conjugate Vaccine



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the organization of such an event, which the community considers to be important from a cultural and economic point of view, public health authorities should consider and anticipate as much as possible the potential sanitary consequences of such a gathering and prepare medical staff for the potential occurrence of unfamiliar diseases.

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Meningococcal Disease in US Military Personnel before and after Adoption of Conjugate Vaccine

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To the Editor: Meningococcal disease in US military personnel is controlled by vaccines, the first of which was developed by the US Army (1–5). In 1985, the quadrivalent polysaccharide vaccine (MPSV-4) was implemented as the military standard. It was replaced during 2006–2008 by the quadrivalent conjugate vaccine (MCV-4). Every person entering US military service is required to receive this vaccine.

Meningococcal disease incidence in active-duty US military personnel, historically far above that in the general population (6), has decreased >90% since the early 1970s, when the first vaccine was introduced (7). Over the last 5 years, incidences in the military and US general populations have become equivalent (8). Here we update previously published data (8) from the Naval Health Research Center's Laboratory-based Meningococcal Disease Surveillance Program of US military personnel. Data-gathering methods and laboratory analyses of samples from personnel suspected of having meningococcal disease have been previously described (8). Incidences were compared by using the New York State Department of Public Health Assessment Indicator based on the methods of Breslow and Day (9).

During 2006–2013 in US military personnel, only 1 of the 28 meningococcal disease cases for which serogroup data are available was not serogroups C or B (8 cases each) or Y (11 cases). During that period, incidence in US military personnel of 0.271 cases per 100,000 person-years did not differ significantly ($p>0.05$) from that of 0.238 in the 2006–2012 age-matched US general population (persons 17–64 years of age) (Centers for Disease Control and Prevention [CDC], unpub. data). During 2010–2013, meningococcal disease incidence in military personnel was 0.174 cases per 100,000 person-years, compared with 0.194 in the age-matched 2010–2012 US population. Among military personnel, only 1 case each occurred in 2011 (serogroup Y) and 2012 (serogroup B), and 3 occurred in 2013 (1 each of serogroups B, C, and Y).

To measure the relative success of the 2 vaccines, we compared incidence among military personnel who

had received MPSV-4 with that of personnel who had received MCV-4. In 2006, MCV-4 was introduced to new recruits. The proportion of military personnel who had received MCV-4, rather than MPSV-4, increased from 6% of the military population (63,000 persons) in 2006 to 64% (930,000) in 2013. By 2013, a total of 99% of new vaccinations were of MCV-4. Overall incidence in personnel receiving MCV-4 was 0.298 cases per 100,000 person-years during 2006–2013, which was lower, although not significantly lower ($p>0.05$), than 0.410 cases per 100,000 person-years in MPSV-4 recipients during 2000–2013.

However, because neither vaccine covers serogroup B, excluding serogroup B cases in the vaccine-related incidence calculations might be more appropriate. Incidence in MCV-4–vaccinated personnel during 2006–2013, excluding serogroup B cases, was 0.183. Specific serogroup data are not available for 2000–2005, so to calculate non-serogroup B incidence during this period, we estimated the proportion of serogroup B cases by examining a range of estimates of serogroup B proportions derived from the true proportions in all 6-year periods during 1995–2012 in the US general population (range 21%–35%; 35% during 2000–2005) (CDC, unpub. data) and during 2006–2013 in US military personnel (range 22%–28%). Adopting (from our estimated range of serogroup B proportions) 21% as the percentage that would have made the MPSV-4–related incidence the highest, MPSV-4–related incidence (i.e., excluding serogroup B cases) during 2000–2013 would have been 0.307, which did not differ significantly from incidence of MCV-4 non-serogroup B cases ($p>0.05$). (Using higher percentages would have pushed the MPSV-4 estimate even closer to the MCV-4 incidence.) The Figure shows pooled incidence for 2000–2013.

Results of these comparisons are subject to several limitations. First, because the relative proportions of the 2 vaccines changed, a differential effect of herd immunity caused by one or the other could have differentially

suppressed rates. Second, along with the decrease in the MPSV-4 population, the average time from vaccination increased relative to the period in which MPSV-4 was given, concomitant with decreasing immunogenicity. Any elevated incidence in the MPSV-4–vaccinated population since 2006 could be associated with time since vaccination. Third, the same factors involved in the decline in incidence in the US general population that began in ≈ 2001 might be at play in the military, confounding the vaccine effects. Fourth, as the rate of vaccine coverage in the US population increased, a higher proportion of recruits might have entered the military already vaccinated; thus, their military vaccination was essentially a booster.

Meningococcal disease incidence decreased during 2000–2013. Our data suggest that cases in MCV-4–vaccinated personnel are similar to those in MPSV-4–vaccinated personnel, regardless of whether the incidence calculation includes cases caused by serogroup B (non-vaccine covered). More extensive study is needed to confirm the relative effects of the vaccines (10). Serogroup B accounted for 5 of the 8 cases during 2012–September 2014), and prevention of disease caused by this serotype remains a challenge.

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The meningococcal disease surveillance in the US military produces a quarterly report, which is available online: <http://www.med.navy.mil/sites/nhrc/geis/Documents/MGCreport.pdf>

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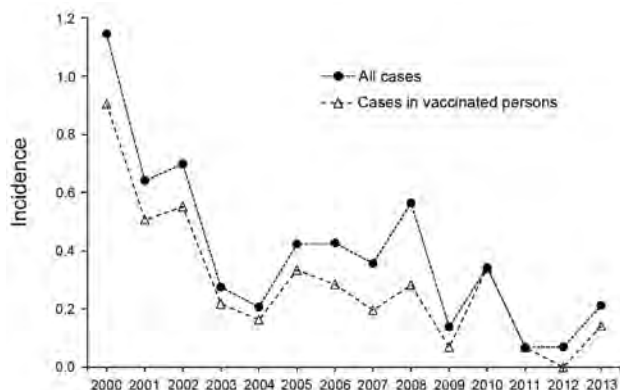


Figure. Meningococcal disease incidence per 100,000 person-years in US military personnel, 2000–2013. Incidence in vaccinated personnel shown assumes that 21% of cases during 2000–2005 were caused by *Neisseria meningitidis* serogroup B.

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Chikungunya Virus Mutation, Indonesia, 2011

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To the Editor: Chikungunya virus (CHIKV) is a single-stranded, positive-sense RNA virus of ≈ 11.8 kb molecules (1) belonging to the family *Togaviridae* and genus *Alphavirus*. Genotypes of CHIKV include Asian, East/Central/South African (ECSA), and West African. CHIKV is endemic to Africa, southern Asia, and Southeast Asia and frequently causes debilitating but nonfatal illness.

CHIKV attracted global attention when a large epidemic on Réunion Island in 2005–2006 spread rapidly to other parts of the world (1). The predominant strain during this epidemic was the ECSA genotype with the A226V mutation of the E1 protein (2), the transmission of which is reported to be facilitated by *Aedes albopictus* mosquitoes (3). The ECSA genotype has been reported to circulate in Southeast Asia, including Malaysia, but not in Indonesia (4). Concern about circulating ECSA strains triggered alerts in 2009, when the Indonesian Ministry of Health reported an increasing number of chikungunya cases (3,529 cases in 2008, 83,756 in 2009) (5). However, only Asian genotypes were detected (4). We investigated recent outbreaks of CHIKV in Indonesia and genotypes of associated CHIKV strains.

After chikungunya outbreaks were reported from 6 districts in Indonesia (Tangerang, Karang Anyar, Ngawi, Jembrana, Mataram, and Kubu Raya), a team from the National Institute of Health and Research Development, Indonesian Ministry of Health, conducted field investigations from April through October 2011. This study received institutional review board approval (KE.01/06/EC/373/2011).

Serum specimens from persons with fever $\geq 38^{\circ}\text{C}$ who provided signed informed consent were tested at the Virology Laboratory, Center for Biomedical and Basic Technology of Health, National Institute of Health Research and Development, in Jakarta. Molecular examination by reverse transcription PCR (RT-PCR) of acute-phase serum specimens, selective for the E1 gene, was performed as previously described (6). Amplicons (330 bp) were sequenced for confirmation. The entire E1 gene of 2 identified ECSA genotypes was sequenced (7). A cladogram was created by using MEGA version 6.06 and the neighbor-joining method (8). The strength of the cladogram was estimated by bootstrap analyses that used 1,000 random samplings. To determine the circulating genotype of CHIKV in Indonesia, we compared these results with other reference sequences in GenBank.

RT-PCR confirmed CHIKV in 28 (26%) of 109 samples from 5 districts: 12 (50%) in Mataram, 8 (47%) in Jembrana, 2 (40%) in Tangerang, 4 (21%) in Ngawi, and 2 (9%) in Kubu Raya. No samples from Karang Anyar were positive for CHIKV. Sequencing analysis revealed the A226V mutant (alanine to valine) ECSA genotype in 2 (7%) specimens (GenBank accession nos. KJ729851, KH729852) and the Asian genotypes (KJ729829–50, KJ729853–56) in 26 (93%) specimens. The Asian genotypes were closely related to those of CHIKV isolated from East Kalimantan, Bandung, Malaysia, and India (Figure).

The 2 cases associated with the A226V mutant ECSA genotype occurred in October 2011 in the Kubu Raya district, West Kalimantan, near the Malaysia border. Because both patients had no history of travel to Malaysia, where outbreaks involving the ECSA genotype had been reported, this finding demonstrates the emergence of the CHIKV A226V ECSA genotype in Indonesia. The 2008 nationwide outbreak of chikungunya in Malaysia proved that A226V mutation enhances transmissibility of CHIKV by *Ae. albopictus* mosquitoes (9). Population movement from this region might contribute to the spread of this virus to Indonesia, which is a concern because of the higher transmissibility of the mutated ECSA strain through the *Ae. albopictus* mosquito vector, which is prevalent throughout Indonesia.

That ECSA genotypes were not found in other districts during this investigation would suggest that this strain was not the source of the 2008–2009 outbreaks in

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